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their meaning because of the special character of the subject in the discussion of which they are employed.

The terms hypocotyl and epicotyl of Darwin, and hyponasty and epinasty of DeVries are objectionable because, being respectively antithetical terms, they are wanting in correlative construction. That is, in their derivation, ἐπί, upon, to, or toward, is made the antithesis of ὑπό, below, or under; whereas ὑπέρ, above, or over is the proper antithesis of ὑπό. Therefore if hypocotyl is used, its antithetic correlative should be hypercotyl; and similarly the correlative of hyponasty should be hypernasty.

Not only are the terms hypocotyl and epicotyl etymologically defective, but their use as originally proposed is not always structurally appropriate. Darwin proposed these terms to indicate the up-growing and down-growing portions respectively of the germinating plantlet, and it is evident from his use of them that he assumed the axis between the opposing portions to be practically identical in position with the points of attachment of the cotyledons. As a matter of fact, however, the cotyledons do not mark any material division in the structure of the plantlet, and the axis referred to is quite independent of their position. In many plants, the bean, for example, the axis is much below the cotyledons and the latter therefore rise above ground as the plantlet grows; while in many other plants, the pea for example, the axis is above the cotyledons, and the latter therefore remain underground. For this inconspicuous, but real, dividing disk between the up-growing and down-growing portions of the plantlet, and also of the mature plant, I have long personally used the term tropaxis, of partially Latinized Greek derivation; and for the parts above and below the axis I have used the adjective terms apotropic, and epitropic respectively.

The terms proposed by Frank, Darwin, DeVries and others have passed into the literature of botany with all their excellencies and imperfections, while my terms apotropism, epitropism and tropaxis have never been published although I have for more than thirty years accustomed myself to their use. I still think they have much merit and therefore offer them for consideration in connection with suggestions for correcting the structure and use of certain terms now generally employed.

CHARLES A. WHITE.

SMITHSONIAN INSTITUTION,  
June 25, 1900.

*LYMPHOSPORIDIUM TRUTTÆ*, NOV. GEN.,  
NOV. SPEC. THE CAUSE OF A RECENT  
BROOK TROUT EPIDEMIC.

In October, 1899, my attention was called to a disastrous epidemic among the brook trout in a Long Island hatchery. The first evidence of the epidemic was seen in May, 1899, when the director picked out a dead fish from one of the ponds and saw that one side was pierced by a clear-cut hole. Thinking the hole due to some bird like a kingfisher, he threw the fish away without further thought. When, however, he found other dead fish with similar wounds, and when the death-rate became noticeably large, an attempt was made to stop the headway of what was then recognized as a disease. Precautionary measures were useless, and during the summer the fish died off at the rate of hundreds per day. Nor did the disease stop until, in December, every fish in the ponds had died.

Investigation begun in October showed the cause of the trouble to be a hitherto undescribed genus of parasitic Protozoa, which I have named *Lymphosporidium truttæ*, belonging to the same class (Sporozoa) as the malaria germ, although the effects of the parasite on the fish are in no way similar to the effect of the malaria-organism in man. Evidences of the disease in the fish were

shown by the sluggish movements and diminished vitality, while many had clear-cut holes or ulcers, as described above. Others appeared with the eyes entirely gone; in others great patches of skin and underlying muscle tissue had fallen out, leaving large irregular pits in the body walls; others still had lost fins or lower jaws, etc.

Upon working out the life-history of the parasite, it was found that spores accumulate in the lymph spaces of the fish and prevent normal nourishment of the tissues, which die and fall out leaving holes in the body-walls. The spores are taken into the digestive tract of the fish—it is not known from where they came originally; in the intestine they give rise to eight sporozoites or germs each of which develops into an adult amoeboid individual not more than .001 inch in length. These adults penetrate the bundles of unstriped muscle cells of the intestine and there become mature. At maturity a spherical spore-forming cyst is formed in the lymph of the fish; here also the spores are liberated, and are then carried to all parts of the body where at different points the accumulations are formed which lead to ulcers.

Two very important points were not determined viz, (1) the origin of the disease which hitherto has probably been unknown, and, (2) the remedy. There was little chance of finding out after October how the disease originated in May, while the extinction of all the diseased fish before the parasite was even discovered effectively headed off experiments with remedial measures.

GARY N. CALKINS.

EMBRYOLOGY OF *LEPAS*. \*

THIS paper was based upon the results of an investigation recently completed, which

\* Abstract of a paper read before the Biological Section of the New York Academy of Sciences, April 9, 1900.

was undertaken with the view of applying the cell-lineage method in an accurate study of the cleavage and the formation of the germ-layers in *Lepas* and other Cirripedes.

The cleavage of *Lepas* is total, unequal, and regular. Stages of 2, 4, 8, 16, 32, and 62 cells are normally formed. Cells of a given generation may anticipate their companions in division, but no second division of such cells takes place before all other cells have completed corresponding cleavages and become of the same generation.

The first cleavage is nearly parallel to the long axis (polar) of the ellipsoidal egg. The egg is divided into an anterior ectoblastic cell and a posterior yolk-bearing macromere. The second cleavage is at right angles to the first, both cells dividing, and from the yolk-macromere is cut off a second ectoblastic cell. The third cleavage is essentially perpendicular to the first two, dividing all the cells, and a third ectoblastic cell is separated from the yolk-macromere, which is now mesentoblastic. Thus by the first, second and third cleavages three protoplasmic cells are separated from the yolk. These three cells contain all the ectoblast and by repeated division they form and extend the blastoderm. The fourth cleavage separates the mesoblast from the entoblast, which is now represented by the yolk-macromere. The 16-cell stage is composed of fourteen ectoblastic cells, which largely surround the entoblastic yolk-cell. The single mesoblast cell lies in the blastoderm at the posterior edge of the blastopore where the entoblastic yolk-cell is still exposed to the exterior. By the fifth cleavage all these cells are divided, the two mesoblastic cells still remaining on the surface. During the sixth cleavage the two mesoblastic cells before dividing sink beneath the blastoderm as it closes over the blastopore. At the same time four cells of the blastoderm, lying at the anterior and lateral edges of the blastopore, divide perpendicularly to